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CORRESPONDENCE

Letters to the Editor

The Challenge of Obesity

Can We Look to the Moon Instead of the Finger?

The multiple challenges of obesity encompass in a simple paradigm what is wrong not only in nutrition but also in the strategies and medical interventions in our society that are aimed at lifestyle change management. This is a "hot" point, which also includes strategies for prescribing healthier and better quality food (1). We also need to enhance more active physical exercise, considering that technology is making life more effortless, while making it more stressful (2). Last but not least, we need to observe with a more open mind the complexity of the problem, because we see only the pale reflection of the light of a still far-off moon.

On the same date as Dr. DeMaria's (3) editorial, another valuable review was published elsewhere (4): "Myths, Presumptions, and Facts About Obesity." However, those investigators skipped several core points. We think that in this field, we can move with greater decisiveness from myths to ideas, going beyond common concepts and dogmas on obesity. In obesity, 2 concepts and concealed dogmas are critical, being presumptions that even go against established facts.

First, obesity is considered invariably ominous for health, trusting that limits, defined by epidemiology, are the gold standards, alone, for healthy weight, obesity, and risks for diseases. Nonetheless, under several conditions, being overweight appears "protective" (the obesity paradox), at least against renal and heart failure (5). This issue is strongly debated (6), but we lack still a good definition of the healthy overweight, if any.

Second, obesity is regarded as an exclusive consequence of an imbalance between food intake and physical exercise, modulated by endocrine and genetic factors. Economists and, obviously, epidemiologists cite environment, but they omit the evidence that environmental infections, and notably adipogenic adenovirus infections in humans, are associated with obesity, being causative factors of obesity and consequent disease in animals (7–9).

In both cases, there is still limited epidemiological information, even based on extensive human investigations in different parts of the world.

As Dr. DeMaria (3) states, the time has come for the medical community to become more organized and proactive in engaging the public and the food industry to emphasize the health hazards associated with obesity: we must seek solutions to the problem without bias and explore these and other neglected fields, such as the quality of nutritional profiles (1), in which considerable evidence is already available.

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Types of Sleep Apnea in Patients With Heart Failure

A Spectrum of Disease Severity or Separate Entities

We read the recently published report by Kasai et al. (1) with keen interest. The investigators performed a study comparing the application of lower body positive airway pressure and its effects on the neck circumference and partial pressure of carbon dioxide in patients with heart failure and sleep apnea. These researchers demonstrated that in all patients with sleep apnea, lower body positive airway pressure led to a reduction in leg volume and to an increase in neck circumference. Interestingly, despite such rostral fluid shift, patients with central sleep apnea (CSA) had a decrease in transpharyngeal resistance, increased minute ventilation and reduced partial pressure of carbon dioxide compared with patients with obstructive sleep apnea, who had opposite findings. We want to sincerely congratulate

the investigators on these important results. However, there are some questions that need to be addressed in future research.

The investigators speculated that pulmonary congestion (although they did not monitor this parameter in the study) and subsequent stimulation of pulmonary irritant receptors might explain why patients with CSA had an increase in minute ventilation and reduced partial pressure of carbon dioxide via increased respiratory drive. Therefore, the question that arises is why more fluid goes to the lungs in patients with CSA compared with patients with obstructive sleep apnea (presumed to be the case because of opposite findings in the study): is it related to a greater fluid volume in cases of more advanced heart failure? It is well known that the prevalence of CSA increases with a greater severity of heart failure (2); thus, the presence of CSA may simply reflect the degree of cardiac dysfunction. Therefore, patterns of sleep apnea may change over time on the basis of the status of cardiac performance.

Alternatively, other mechanisms yet to be elucidated may be responsible for greater pulmonary congestion in patients with CSA. Fluid overload and pulmonary congestion may explain why patients with renal disease have a greater prevalence of CSA compared with the general population (3). Therefore, it is important to study whether pulmonary congestion and stimulation of pulmonary irritant receptors will explain the different response to rostral fluid shift in patients with CSA and obstructive sleep apnea (4).

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Reply

Types of Sleep Apnea in Patients With Heart Failure: A Spectrum of Disease Severity or Separate Entities

Drs. Mirrakhimov and Mirrakhimov ask why more fluid would be redistributed in the lungs by application of lower body positive pressure (LBPP) in patients with heart failure with central sleep apnea (CSA) than in those with obstructive sleep apnea, thus stimulating pulmonary vagal irritant receptors that provoke hyperventilation. They ask further whether this might be due to a greater degree of volume overload in those with CSA.

In fact, we stated in the report that such differing responses may be related to differing cardiac loading conditions (1). For example, previous studies have shown that nonchemical pulmonary irritant receptor stimulation by pulmonary congestion provokes hypocapnia (2), that in patients with heart failure, partial pressure of carbon dioxide is inversely proportional to left ventricular filling pressure (3), and that reducing left ventricular filling pressure was associated with attenuation of CSA (4). In our study, we observed that compared with obstructive-dominant patients, central-dominant patients had a much greater degree of mitral regurgitation, as well as higher right ventricular systolic pressure and levels of N-terminal pro–B-type natriuretic peptide, all of which are indicative of greater left ventricular filling pressures and suggestive of greater pulmonary congestion (5). Accordingly, for a similar fluid shift from the legs into the heart in response to LBPP, pulmonary congestion with stimulation of vagal irritant receptors, a consequent increase in ventilation, and a decrease in partial pressure of carbon dioxide would more likely occur in patients with higher than in those with lower left ventricular filling pressures. The significant relationships between N-terminal pro–B-type natriuretic peptide levels and indicators of increased respiratory drive in response to LBPP further support this concept.

Drs. Mirrakhimov and Mirrakhimov also speculate that the type of sleep apnea could change over time in response to alterations in cardiac function. We agree, and in fact cited our previous work, in which we demonstrated that the predominant type of sleep apnea could shift from obstructive sleep apnea to CSA over time, in association with a deterioration in cardiac function and a decrease in partial pressure of carbon dioxide, and vice versa (6,7). We also agree that fluid overload and pulmonary congestion may explain the higher prevalence of CSA in the renal failure population than in the general population.

Finally, we concur with Drs. Mirrakhimov and Mirrakhimov that future studies would ideally directly quantify lung water or left ventricular filling pressure in response to interventions such as LBPP and take us a step closer to defining the mechanism for the hyperventilatory response to LBPP observed in our patients with heart failure with predominantly CSA.

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